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## NEWCASTLE DISEASE IN SHAGS AND CORMORANTS AND ITS SIGNIFICANCE AS A FACTOR IN THE SPREAD OF THIS DISEASE AMONG DOMESTIC POULTRY

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### INTRODUCTION

Beaudette (1943), in his comprehensive review of the literature on Newcastle disease, refers to field reports of the existence of this disease among swans and parrots in the Dutch East Indies, mayas (a species of martin) in the Philippine Islands, and sparrows in India. It is also known (Biester & Schwarte, 1948) that other species of wild birds are susceptible to inoculation with virus, and there are reports describing the disease in Chinese and silver pheasants, Californian quail, Hungarian and chukar partridges. Certain species of doves and the common pigeon are also susceptible to artificial infection with the virus. Levine *et al.* (1947) described an outbreak which occurred in ring-necked pheasants reared intensively on a farm where Newcastle disease had been confirmed a few months earlier in turkeys ; direct transmission between the two species was not established. There are only three references, however, to the actual isolation of Newcastle disease virus from wild birds : Wilson (1950) describes the transmission of the disease to laboratory fowls by inoculation of a tissue emulsion from a gannet, *Sula bassana*, picked up on the beach of one of the Orkney Islands during investigations into the origin of the same series of outbreaks to which reference is made in this paper. Gillespie *et al.* (1950) in the U.S.A. isolated Newcastle disease virus from a 12-day-old starling, *Sturnus vulgaris*, which had fallen from its nest ; there was no evidence that the parent birds were infected. Ingalls *et al.* (1951), also in the United States, described nervous symptoms in and the subsequent isolation of virus from a great horned owl, *Bubo virginianus virginianus*, which had been presented to a zoo six weeks previously by a local farmer.

The demonstration of anti-haemagglutinins for Newcastle disease virus in sera collected from wild shags shot in the vicinity of islands in the Hebrides, where outbreaks of Newcastle disease had been confirmed, and the subsequent isolation of virus from the bone marrow of certain of these birds was of interest because, although it did not provide an origin for the initial outbreaks, it presented a possible explanation for some of the spread of the disease which occurred in that area.

### HISTORY OF THE OUTBREAKS

Newcastle disease was confirmed among domestic poultry in the Orkney Islands in September, 1949. The origin of this series of outbreaks was never clearly established and there was, in fact, some doubt as to whether the original case was in the Orkneys. During the subsequent enquiries, an outbreak was confirmed on the island of South Uist in the Hebrides and the history suggested that disease had existed there about the same time that its presence was confirmed in the Orkneys. A possible source of the primary infection in these outbreaks was thought to have been the deposition of infected carcases or viscera on the sea shore after their disposal overboard from visiting yachts or other shipping. Spread of the disease to other islands round the coast of Scotland and to many of those in the Outer Hebrides was considered to have been due to the flow of tides and currents, which could have carried infected carcases from one island to another after they had been thrown into the sea by the islanders themselves. Newcastle disease virus was isolated at Weybridge from the bone marrow of the putrefied carcase of a fowl picked up on the beach of one of the Orkney Islands, and domestic fowls in the coastal villages on these islands were often found scavenging alongside sea birds among the debris washed up on the beaches.

Not all the outbreaks which occurred in this area during the autumn of 1949 could be explained in this way, however, and the widespread distribution of the disease on both the east and west coast of Scotland led to the consideration of the possibility that wild sea birds might be responsible for spread of some of the infection. During the investigation of certain outbreaks it was discovered that the shag, *Phalacrocorax aristotelis*, or in Gaelic, *Scarbh' beag*, is shot every year in considerable numbers for human food by the inhabitants of many of the Western Islands and also on the mainland of Inverness-shire. This bird is known locally as the scart or lesser cormorant. Attention was first directed particularly to this species when two outbreaks of Newcastle disease were confirmed on the island of Gigha, Argyllshire, among poultry whose owners were in the habit of shooting shags for their own consumption. After dressing the carcases it was customary to throw the viscera and unwanted trimmings to the poultry. Newcastle disease virus was subsequently isolated from the bone marrow of the long bones of some of the shags shot by the owner of the stock in one of these outbreaks. Subsequently several other outbreaks in the Outer Hebrides were confirmed among poultry to which the viscera of shags had been fed.

These investigations also revealed that the cormorant, *Phalacrocorax carbo*, is shot for human consumption in some areas, although its flesh is not considered so palatable as that of the shag.

With the co-operation of the field veterinary officers of the Animal Health Division of the Ministry, a number of shags and cormorants were shot in the vicinity of islands where outbreaks of disease had been confirmed and blood samples were collected from the recently dead or dying birds and sent to Weybridge for haemagglutination-inhibition (H.I.) test.

Whenever possible, long bones were also sent from the dead birds so that an attempt could be made to isolate virus from the bone marrow. During the same period a number of other wild birds were shot and the carcases, together with blood samples if obtainable, were collected for examination.

### Haemagglutination-Inhibition Tests on Sera of Wild Sea Birds

The results of the H.I. tests carried out on wild birds during this investigation are summarised in Table I.

Table I

| Species of bird | Number of samples examined | Haemagglutination-inhibition titre Units 5–20 | 40 | 80 | 160 | 320 | 640 | 1,600 |
|---|---|---|---|---|---|---|---|---|
| Shag, *P. aristotelis* ... | 54 | 29 | 7 | 5 | 4 | 8 | 0 | 1 |
| Herring gull, *Larus argentatus* ... | 11 | 11 | | | | | | |
| Blackheaded gull, *Larus ridibundus* ... | 3 | 3 | | | | | | |
| Great Northern diver, *Colymbus immer* ... | 2 | 1 | 1 | | | | | |
| Oyster catcher, *Haematopus ostralegus occidentalis* ... | 1 | | 1 | | | | | |
| Guillemot, *Uria aalge* | 1 | 1 | | | | | | |
| Hooded crow, *Corvus cornix* ... ... | 2 | 2 | | | | | | |

The technique used in carrying out these tests was a slight modification of the method described in the supplement to the U.S. Department of Agriculture's recommendations for the diagnosis of Newcastle disease (1946). This technique has been adopted as a routine test for the diagnosis of suspected Newcastle disease since the re-introduction of the disease into England (Gordon & Asplin, 1947). Experience gained when testing thousands of blood samples from fowls, bantams, turkeys, ducks and geese has shown that an H.I. titre of 320 units or more almost invariably indicates the presence of specific antibody in the serum of a bird of these species. Titres of 80 to 160 H.I. units, particularly when accompanied by higher titres in other samples from the same source, normally indicate recent infection. Titres of 40 units may be of no significance in individual sera but, if there are higher titres in other samples from the same group, they sometimes indicate recent infection.

The total of 54 blood samples from shags included a small number of cormorants. The exact number cannot be shown because at this time there was a misunderstanding between the laboratory and the field staff as to the identification of these two species owing to the use of local names. The sera showing an H.I. titre of 320 units included, however, at least one cormorant. The birds, from which blood samples showing a significant titre were collected, were all shot in the vicinity of confirmed outbreaks on the islands of Harris, Lewis and Tiree.

### The Examination of the Carcases of Wild Birds for the Detection of Virus

The legs of birds which had been shot were despatched to the laboratory. The bone marrow was collected with sterile precautions from the femurs and a crude saline emulsion prepared using Griffiths grinding tubes. Whenever a positive or doubtful H.I. titre had been demonstrated in a bird from which leg bones also had been received, a separate emulsion was prepared. When blood samples were not available or when H.I. titres were negative, emulsions were prepared from the bone marrow collected from one up to groups of eight birds. Each emulsion was divided, one being used, as soon as possible after collection, for intramuscular injection into susceptible fowls, the other being tested for sterility, and filtered when necessary through a gradocol membrane, prior to the inoculation of chicken embryos.

When starlings were killed for examination the whole carcase was sent to the laboratory, and the emulsions were prepared from the livers using the same technique.

The results of these examinations are summarised in Table II.

Table II

| | Species of bird: Shag, *P. aristotelis* | Herring gull, *L. argentatus* | Starling, *Sturnus vulgaris* | Guillemot, *U. aalge* | Northern diver, *C. immer* |
|---|---|---|---|---|---|
| Number of birds received ... | 60 | 4 | 26 | 2 | 2 |
| Number of emulsions tested for evidence of virus | 26 | 4 | 5 | 2 | 2 |
| Number of positive isolations ... | 6 | 0 | 0 | 0 | 0 |

In addition, one bird of each of the following species was examined with negative results : Hooded crow, *C. cornix* ; fulmar petrel, *Fulmarus glacialis* ; oyster catcher, *H. ostralegus occidentalis* ; red-throated diver, *Colymbus stellatus* ; shearwater, *Puffinus puffinus* ; rockdove, *Columba livia.*

Four of the six positive isolations from shags were from birds from which blood samples had been tested previously ; one had shown an H.I. titre of 320 units, and one of 160 units but the titre of the other two birds had been less than 20 units. The two remaining positive isolations were from the bones of two groups, each of five birds, obtained from a premises where Newcastle disease had already been confirmed in the poultry ; blood samples from these 10 shags were not obtainable.

Again the total of 60 shags included a few cormorants, *P. carbo*, but it was later established that in no case had a positive virus isolation been obtained from this species.

### Identification and Pathogenicity of the Viruses

The six strains of virus isolated from shags were serologically and immunologically identical to the virus of Newcastle disease.

There was considerable variation in the pathogenicity of the six strains when tested by the inoculation of susceptible fowls. Only three of them produced symptoms and *post-mortem* lesions of Newcastle disease ; the other three strains produced an H.I. titre in the sera of the inoculated fowls but without causing recognisable symptoms of Newcastle disease. All six strains killed baby chicks four to five days old in from three to five days after inoculation.

### Discussion

In assessing the significance of the isolation of Newcastle disease virus from wild sea birds, several points must be considered. The reports in the literature indicate that it is probable that most species of birds are susceptible to infection with this virus, if brought into sufficiently close contact with it. Both the shag and the cormorant are fish-eating birds and not scavengers along the sea shore ; they are seldom seen in close association with man and his dwellings. They do, however, congregate on rocks and small islands alongside large numbers of the scavenging gulls, some of which may have fed on Newcastle disease infected carcases : this would seem to be the most likely source of infection. An alternative theory put forward by one of the veterinary

officers engaged in the investigation of these outbreaks was that cormorants and shags might have become infected by mistaking the floating intestines of poultry for a fish or an eel. This opinion was further supported by a local ornithologist. There was no reliable evidence to suggest that there was any undue mortality or illness among sea birds during or before the occurrence of Newcastle disease outbreaks in fowls. Judging from experience with domestic ducks and geese, it is possible that disease in the wild birds may have been comparatively mild. It is unlikely that this potential source of infection would have been discovered if shags and cormorants had not been shot for human consumption and if the coincidental occurrence of Newcastle disease in domestic poultry, which had received uncooked viscera from the carcases, had not been noted. Whether or not disease still exists among the wild shags and cormorants in the Hebrides and round the Scottish coast is not known, but in any case very few outbreaks of Newcastle disease have been confirmed in this locality since the major epidemic of 1949, despite the fact that presumably both species were shot for human consumption during the autumn of 1950. It is known, however, that the intensive propaganda put out by the Animal Health Division of the Ministry of Agriculture had some effect on the islanders and they may therefore have exercised more care in the disposal of the unwanted portions of the birds which had been shot.

Prior to 1947, Newcastle disease had been unknown in Great Britain since the single outbreak occurring in 1933 and therefore there is not likely to have been any other source of infection to wild sea birds in the North Sea area until the first outbreaks occurred in the Outer Hebrides and Orkney Islands in September, 1949. The widespread epidemic occurring in England, subsequent to February, 1947, had been confined very largely to the London area and to the large industrial centres. Subsequent to the outbreaks in Scotland, Newcastle disease was diagnosed in both Southern and Northern Ireland. The origin of the outbreaks in Northern Ireland was never established but circumstantial evidence incriminated wild sea birds. In that area the first outbreak was on a small farm within sight of the sea and on a part of the coast which was only about 45 miles distant from the Scottish coast and islands where outbreaks had occurred a few weeks earlier. From the point of view of substantiating this theory, it was disappointing that the only other confirmed isolation of Newcastle disease virus from sea birds during the investigation of these outbreaks was that reported by Wilson (1950) from a gannet. The gannet, like the shag and cormorant, is not a scavenger and would not normally have any contact with domestic poultry. On the other hand, it is possible that, if larger numbers of scavenging gulls had been examined, evidence of infection in other species might also have been established.

The evidence presented in this paper is considered sufficient to justify the conclusion that shags and cormorants became infected with Newcastle disease during the 1949 epidemic in Scotland and were probably an important factor in the spread of disease to domestic poultry under the particular circumstances which have been described.


### Summary

The presence of anti-haemagglutinins of Newcastle disease virus is reported in the blood serum of 18 out of a total of 54 samples collected from shags, *Phalacrocorax aristotelis*, and cormorants, *Phalacrocorax carbo*. Subsequently, six strains of a virus, serologically and immunologically identical to that of Newcastle disease, were isolated from 26 emulsions prepared from the bone marrow of shags.

The significance of the presence of this virus in wild shags and cormorants, as a potential source of infection to domestic poultry, is discussed.



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## NOTES ON THE SEMEN OF STERILE AND SUB-FERTILE BULLS


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The secretory activity of the accessory reproductive glands is dependent on the androgenic function of the testis and any alteration in accessory gland function will have a corresponding effect on the seminal fluid (see Anderson, 1945). Mann (1946a, b) established that fructose is a constituent of seminal fluid. The main source of fructose in the bull is the seminal vesicles, but it is also found in the ampullae of the vas deferens (Mann, 1946b). The formation of fructose in the accessory organs is initiated, maintained and controlled by the testicular hormone and characteristic fluctuations can be evoked in response to treatment by testosterone (Mann & Parsons, 1947). The *p*H, buffer capacity and specific gravity of seminal fluid depend on accessory gland secretions, probably chiefly the seminal vesicles (Anderson, 1949). These qualities of seminal fluid are related in that the more acid the seminal fluid the better the buffer capacity, and the higher the specific gravity and the fructose content (Ander-

son, 1946a, b; 1951). Clearly, these qualities of seminal fluid can be used to detect deviations from normal function in the accessory reproductive organs, particularly the seminal vesicles. Over a period of years it has been possible to examine some of these criteria in sterile and other bulls.

## Material and Methods

The material consisted of 11 bulls affected with varying degrees of clinical epididymitis, which is the diagnostic feature of what is probably the most important cause of bovine sterility in Kenya (Anderson, 1949; Hudson, 1949), one bull with orchitis and one bull with morphologically abnormal spermatozoa.

Semen was collected in the usual way with the artificial vagina.

The methods used for determining the sugar content, the buffer capacity and the specific gravity have already been described (Anderson, 1946a, b). The *p*H was determined with the Beckman *p*H meter, as was the change in *p*H after incubation of semen for one hour at 37° C.

## Results

Bilateral Epididymitis.—Eighteen semen specimens were collected from four bulls which clinically had bilateral epididymitis. Spermatozoa were absent from the semen of two bulls (Nos. 3 and 4) and were either absent or present in very small numbers (1·0 to 4·0 × $10^7$ sperm per 1 ml.) in the other two bulls (Nos. 1 and 2). Only in one specimen, the first from No. 2, was there very slight motility; all the others were completely immotile. The *p*H of all specimens was alkaline. The fructose content for No. 1 averaged 152 mg. per 100 ml. semen (range 0 mg. to 376 mg.); for No. 2, 28·4 mg. (range 0 mg. to 116 mg.); and for No. 3, 205 mg. The buffer capacity (B.C.) for this specimen from No. 3 was 3·14 and the specific gravity (S.G.) 1·0268.

Unilateral Epididymitis.—There were five bulls with clinical unilateral epididymitis.

*No.* 5. Three specimens gave a motility of 70 per cent. to 90 per cent., density 5·3 × $10^8$ sperm per 1 ml., *p*H 6·83. There was no change in the clinical picture over the period of a year, when two specimens gave a motility of nil and 70 per cent., density 5·3 and 5·0 × $10^8$ sperm per 1 ml., and *p*H 7·93 and 7·76 respectively.

*No.* 6, pedigree Aberdeen Angus. The motility for six specimens over nine months was 0 per cent. to 100 per cent. (average 60 per cent.), *p*H 6·72 to 7·70. Two specimens at the end of this period had a motility of 40 per cent. and 60 per cent., *p*H 6·73 and 6·93, *p*H change + 0·38 and − 0·10, and fructose content 584 mg. and 564 mg. per 100 ml. semen respectively.

*No.* 7, grade Shorthorn. The clinically unaffected testicle was removed in October, 1941. Eight specimens over the following 10 months had a motility of 0 per cent. to 50 per cent. (average 25 per cent.), a small volume—1·5 ml. to 2·5 ml., density 1·9 to 19·0 × $10^8$ sperm per 1 ml., *p*H 7·02 to 7·55 (average 7·20) and *p*H change + 0·18 to −0·78 (average −0·29). The *p*H, *p*H change and motility improved towards the end of this period.

*No.* 8, pedigree Friesian. Ten specimens from this bull, over a period of 10 months, after removal of one testicle in October, 1941, had a motility of 0 per cent. to 70 per cent. (average 40 per cent.), density 1·6 to 4·5 × $10^8$ sperm per 1 ml., *p*H 7·23 to 7·99 (average 7·60) and *p*H change + 0·16 to −0·41 (average −0·21). Towards the end of this period motility was 60 per cent. and the *p*H change −0·45, but the *p*H remained high—7·60. From June to September, 1942, 66 inseminations with semen from this bull resulted in 12 conceptions.

*No.* 9, pedigree Friesian. This bull was first tested in February, 1941, when three specimens were collected. The volume was small, about 2 ml., the motility was 10 per cent., 30 per cent. and 100 per cent., density 2·9 to 12·7 × $10^8$ sperm per 1 ml., and fructose was absent in all specimens. Eight further specimens up to the end of March gave a similar picture, but a maximum of up to 124 mg. fructose per 100 ml. semen was found.

In the middle of June a marked orchitis was observed on the right side, at which time the semen was of good quality—motility 80 per cent. to 100 per cent., density 5·2 to 5·4 × $10^8$ sperm per 1 ml., and fructose 212 mg. and 240 mg. Three months later the maximum motility was 70 per cent. and fructose was absent in two specimens. The clinically affected testicle was then removed. Two months later the motility was 90 per cent., density 11·2 × $10^8$ sperm per 1 ml., and the *p*H 7·15. Six specimens in 1942 had a motility of 40 per cent. to 80 per cent. (average 65 per cent.), density 3·1 to 9·6 × $10^8$ sperm per 1 ml., *p*H 7·03 to 7·82 and the *p*H change −0·29 to −0·56. From March to December, 1942, 64 inseminations resulted in 16 conceptions.

Up to March, 1943, of four specimens the motility was 60 per cent. to 90 per cent. (average 80 per cent.); density 6·9 to 9·3 × $10^8$ sperm per 1 ml., *p*H 6·92 to 7·79 and the *p*H change + 0·17 to −0·39. One of these specimens with a *p*H of 7·35 and a *p*H change of +0·17 was used to inseminate two cows, both of which conceived.

The tunica vaginalis of the affected testicle was strongly adherent to the epididymis and testis. The head, body and tail of the epididymis was enlarged and fibrosed, and thick, white purulent material was present in the body and head. Smears from the testis and epididymis showed numerous polymorphonuclear leucocytes, much cellular debris and no spermatozoa.

"Early" Epididymitis.—Neither of the two bulls, pedigree Friesians, had clinical epididymitis, but as they were in a herd in which the disease was widespread, and because the semen resembled typical early cases of epididymitis, they were regarded as being probably in the early stages of the disease. The bulls were slaughtered before clinical evidence of the disease developed.

Eleven specimens were collected from No. 10. Seven showed no motility and the other four, all second collections, 10 per cent. to 60 per cent. motility. Motility became progressively poorer over the month of observation. Density was 6·0 to 8·5 × $10^8$ sperm per 1 ml., and the semen was alkaline throughout. Fructose varied from 0 mg. to 84 mg.

Thirteen specimens were collected from No. 11, of which six showed no motility and the others 10 per cent. to 60 per cent. Density was low—1·0 × $10^7$ to 4·4 × $10^8$ sperm per 1 ml. The semen was alkaline and the fructose content varied from 92 mg. to 608 mg. (average 334 mg.).

Orchitis.—No. 12, pedigree Ayrshire. When first seen in July, 1943, this bull had a marked orchitis on the left side, which had apparently been developing for three weeks previously. The epididymis was not affected. The last fertile service was on May 29th. Three services from July 13th to 17th were not fertile. A blood test for contagious abortion was negative.

Two semen specimens on July 24th had motility 10 per cent. and 70 per cent., density 0·9 and 1·4 × $10^8$ sperm per 1 ml., *p*H 7·53 and 8·48, *p*H change −0·10 and −0·18 and buffer capacity 2·88 and 2·37 respectively. On October 12th

the swelling was larger and firmer and the semen showed —motility 50 per cent. and nil, density 2·5 and 0·3 × $10^8$ sperm per 1 ml., *p*H 7·60 and 8·23, *p*H change −0·27 and −0·23 and B.C. 2·08 and 3·57. On November 24th the swelling was somewhat reduced and the tail of the epididymis could be palpated; spermatozoa were absent from the semen and the *p*H was 8·12. The affected testicle was removed on December 6th. Fifteen weeks later the motility was 100 per cent., density 13·7 × $10^8$ sperm per 1 ml., and the *p*H 7·62 (March 28th, 1944). Details of the service record were not available but the bull was known to be fertile and was in use for some years.

Bull with Morphologically Abnormal Spermatozoa.—No. 13, pure-bred Jersey, four years old. This bull was used solely for artificial insemination and proved sterile. Eight tests were made during April and May, 1950. The motility was only moderately good—average 60 per cent. (range 40 per cent. to 90 per cent.); sperm density 2·7 to 8·2 × $10^8$ sperm per 1 ml., *p*H 6·76 to 6·89; fructose content of seminal fluid averaged 574 mg. per 100 ml. (range 473 mg. to 702 mg.); percentage fructolysis, 0 to 13·9 (average 4·3 per cent.); fructolysis index (Mann, 1948a, b) 0 mg. to 0·91 mg. × $10^9$ sperm per one hour. Primary abnormalities in the spermatozoa averaged 41 per cent. and secondary abnormalities 22 per cent. (Blom's classification, 1948). The following tests were made on two specimens only:—*p*H change −0·21 and −0·52; B.C. 2·00 and 2·46 and S.G. 1·0257 and 1·0272.

The factors relating to the seminal fluid-*p*H, fructose content, B.C. and S.G. fall within the normal range, but a high proportion of spermatozoa were morphologically abnormal and of reduced metabolic activity.

## Discussion

*p*H.—The semen of fertile bulls is typically acid and that of sterile bulls usually alkaline. The *p*H of typical cases of epididymitis varied from 6·75 to 8·39, and only 6 per cent. of specimens had a *p*H below 7·00 (Anderson, 1942). The results are confirmed by the present data. The four bulls with bilateral epididymitis, as well as the two bulls with "early" epididymitis always had alkaline semen. Bulls 5 and 6, which had recovered from an acute attack of epididymitis, leaving one testis and epididymis normal, had an average *p*H of 7·41 and 7·05, associated with a fairly good motility (60 per cent.). No. 9 had an average *p*H of 7·36 associated with a good motility (70 per cent.) after removal of the affected testicle; only 2 of 10 specimens were of *p*H less than 7·00. Bulls 7, 8 and 12 also after removal of the affected testicle had an average *p*H of 7·20, 7·60 and 7·62 and a motility of 25 per cent., 40 per cent. and 100 per cent. respectively.

In semen from fertile bulls, the lower the *p*H the greater is the concentration of sperm and the higher the motility and *vice versa* (see Anderson, 1945), but about 10 per cent. of ejaculates have a *p*H somewhat above the average—6·91 to 7·20, with a fairly good motility (average 76 per cent.). Bulls 5, 8, 9 and 12 which had recovered from epididymitis, or had had the clinically affected testicle removed were exceptional in that a fairly good motility was associated with a rather high *p*H.

*Buffer capacity and specific gravity.*—The normal B.C. and S.G. is 1·84 and 1·0377 (Anderson, 1946a). A poor B.C. (*i.e.*, higher than the normal figure) and a low S.G. have been noted previously in epididymitis. In the present data, bull No. 3 with bilateral epididymitis had a poor B.C. (3·14) and a low S.G. (1·0368). Bull No. 12 with orchitis also had a poor B.C. (average 2·73).

*Fructose.*—Mann (1948b) remarked that semen samples with a notoriously low fructose content were those from animals with a poor fertility record, and cited five cases of bulls in which the fructose content of semen was below 300 mg. per cent.: (1) two bulls with necrospermy, with fructose ranging from 160 mg. to 190 mg., (2) two infertile bulls with semen containing less than 100,000 sperm per c.mm., and 248 mg. to 334 mg. fructose and (3) one bull with normal sperm density and motility, which sperm died rapidly on storage—fructose content 30 mg. to 360 mg.

The sugar content of bull semen has been found to average 470·5 ± 11·7 mg. (S.D. = 181·4 mg., Anderson, 1946b), and Mann (1946a, b) has observed rather higher amounts. The fructose content of bulls Nos. 1, 2 and 3, namely, 152 mg., 28 mg. and 205 mg., is considerably below the normal level. Bull No. 9 had a fructose content of 0 mg. to 240 mg. three months before any clinical symptoms developed, and three months after orchitis and epididymitis developed the fructose content was nil. Although the fructose content of No. 9 was extremely low, the density of sperm was fairly high and the motility good, if variable, and the bull was fertile. On the seven occasions on which two or more ejaculates were collected in succession, the motility and density improved progressively, but there was no alteration in the low fructose level. Mann (1948b) noted that when a bull is subjected to an "exhaustion test," in which semen is collected at frequent and short intervals, there was a marked fall in sperm density but the fructose content maintained a high level throughout. When first tested, the semen of this bull contained mucus, which yielded a clot on deproteinisation with zinc hydroxide, when determining the sugar content. Of the bulls with "early" epididymitis, No. 11 had a variable fructose content and No. 10 a very low fructose content. The fructose content for No. 6 was normal, conforming with the acid *p*H.

*p*H *Change.*—The mean *p*H change in semen after one hour's incubation at 37° C. was found to be −0·302 ± 0·027, for an average density of 6·7 × $10^8$ sperm per 1 ml. The average *p*H change for bulls 7, 8 and 9 after removal of one testicle was −0·29 (density 7·8 × $10^8$ sperm per 1 ml., motility 25 per cent.); −0·21 (density 3·4 × $10^8$ sperm per 1 ml., motility 40 per cent.); −0·20 (density 7·2 × $10^8$ sperm per 1 ml., motility 70 per cent.) respectively. The low *p*H change in these bulls may have been affected by the lower motility and in No. 8 by the low density.

*General.*—An alkaline *p*H is associated with a poorer buffer capacity, a lower specific gravity and a lower fructose content (Anderson, 1946a, b; 1951). This is borne out by No. 3 for *p*H, B.C., S.G., and fructose content, by Nos. 1, 2, 9, 10 and 11 for fructose and *p*H, and by No. 12 for B.C. and *p*H.

It is not clear whether the changes in the seminal fluid can be attributed primarily to deficient secretion of male sex hormone, or secondarily to failure of the secretory structure of the seminal vesicles and ampullae to respond to male sex hormone, or to both causes, for the testes and accessory glands undergo pathological changes in this disease.

Mann (1948a) noted that azoospermic and necrospermic semen cannot utilise fructose and that a reduced rate of fructolysis is found in sperm of low fertility from subfertile and infertile bulls. Bull No. 13 provides an example of a sterile bull with a high percentage of abnormal spermatozoa with a low rate of fructolysis.

(*Concluded at foot of col. 1, p. 736*)

# VETERINARY PHARMACY*

BY

F. W. ADAMS, B.Sc., PH.C., A.R.I.C.,

SECRETARY AND REGISTRAR, THE PHARMACEUTICAL SOCIETY OF GREAT BRITAIN

I must make it clear at the outset that I do not speak on this subject as an authority. As an official of the Pharmaceutical Society of Great Britain, however, I have in recent years been closely concerned with the professional and political aspects of veterinary pharmacy and can perhaps give some information on these aspects which will be of interest to the Assembly.

So far as I am aware, this is the first time that veterinary pharmacy has been the subject of discussion within the International Pharmaceutical Federation. I have examined all the Bulletins issued since the Federation was founded in 1912 but can find no reference to any article or paper on the subject. Its inclusion in the agenda of this Assembly is to be welcomed as an indication of concern with an increasingly important field of pharmaceutical activity.

Some account of the position of the practice of veterinary medicine and surgery in Great Britain may form a useful introduction to a consideration of veterinary pharmacy.

Until 1881 there were no laws affecting veterinary practice. Anyone could practise veterinary medicine and surgery, and side by side with an organised veterinary profession which had existed since 1844, when the Royal College of Veterinary Surgeons was established by Royal Charter, persons unqualified in veterinary medicine and surgery carried on veterinary practice. These ranged from those who made it their sole means of livelihood and practised all branches of the veterinary art to those who became involved to some extent in veterinary activities through selling veterinary drugs and medicines. In 1881 an Act of Parliament was passed to enable persons desiring the aid of a veterinary surgeon to distinguish between qualified and unqualified persons. A register of veterinary surgeons was established and unregistered persons were not allowed to use certain titles or descriptions or to recover in court any fees for veterinary treatment. Admission to the register in the future was to be only by way of membership of the Royal College. No substantial change in the position took place until 1948, when an Act of Parliament was passed which made important changes in, and strengthened the position of, the veterinary profession.

Although the Act of 1881 was a step in the right direction it did not prevent unqualified practice and for this and the following reasons circumstances remained unfavourable to the veterinary profession, retarded recruitment to it and resulted in insufficient use being made of its services. These reasons were the decline in agricultural prosperity following the importation of cheap foodstuffs which set in during the later years of the last century and the failure to adjust production methods to meet the new circumstances, the lack of public and official interest in the promotion of animal husbandry, and the inadequate appreciation by many farmers of the value of the services of qualified veterinarians.

There has in recent years been a growing realisation of the need for a national policy of developing food production in Great Britain to reduce reliance on overseas supplies and to make the most of the natural resources of the country. Particularly during and since the last war substantial progress in this direction has been made. Agriculture and animal husbandry are being fostered by Government measures in various ways with the object of bringing these branches of the national economy up to date and to overcome the neglect of past years. Farming and stock-keeping is being put on a more scientific basis and in this connection veterinary science and its application in improving the quality and health of livestock has received increasing attention from official quarters. Following Government enquiries into the needs of veterinary education and the effect of unqualified veterinary practice, steps have been taken to extend and develop facilities for veterinary education and to put an end to unregistered practice. The Veterinary Surgeons Act 1948 makes veterinary practice a "closed" profession and only registered persons may now engage in it. All future entrants to the profession must take the examinations of the Royal College of Veterinary Surgeons or possess a university degree in veterinary medicine and surgery recognised by the Royal College.

To those familiar with the limited legal restrictions under which drugs and medicines for human use are prepared and distributed in Great Britain it will come probably as no surprise to learn that there is even less control over veterinary medicines. No qualification is required to prepare veterinary medicines or to sell them to farmers or stock-keepers, except in the case of narcotic drugs and of penicillin and other antibiotics subject to similar conditions. These can be supplied only by medical and veterinary practitioners and pharmacists. Apart from this, only in the case of preparations containing substances classed by law as poisons and sold to owners of pets and other domestic animals is a qualification necessary, in which case the supply must be obtained from a pharmacy. Even this requirement is not absolute, as under certain conditions persons other than pharmacists who are engaged in the actual manufacture of animal medicines may sell their preparations to the public.

In the case of preparations containing the more powerful poisons, whether sold to farmers or domestic animal owners, the purchaser must be known to the seller as a person to whom the preparation may properly be sold and a record made of the transaction by the seller. Preparations containing poisons must have the name and quantity of the poison stated on the label and if made up ready for use, the label must bear the words: "Poison, for animal treatment only." Ribbed or grooved bottles must be used for

* Presented to the General Assembly, International Pharmaceutical Federation, Rome, September, 1951.

SUMMARY

An account is given of various criteria, including *p*H, buffer capacity, specific gravity, fructose content and *p*H change in the semen of bulls with epididymitis, orchitis and morphologically abnormal spermatozoa.

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liquid preparations for external use. That, broadly, is the extent of the requirements.

As to the quality of veterinary drugs and medicines, there are no established standards for these. It is at least doubtful whether there is any legal control of the quality of veterinary drugs and although it is conceivable that the sale of unsatisfactory material might result in a prosecution, I have not been able to discover that anyone has been prosecuted for supplying veterinary drugs of inferior quality.

The composition of veterinary medicines is not required to be disclosed and there is no regulation of the claims made for veterinary medicines or of their advertising.

Taking into consideration the volume and nature of veterinary medicines, the legal restrictions taken altogether do not represent any effective control of the preparation and distribution of veterinary medicines as a class and place virtually no barrier in the way of undesirable practices. Thus the user of animal medicines has to rely almost entirely upon the knowledge and integrity of the individual makers and suppliers of such medicines in obtaining material of the nature and quality needed.

Veterinary products are made and supplied by various classes of people. Pharmacists in country towns, particularly market towns, have traditionally supplied the farmer who treats his livestock himself with the drugs and preparations he needs in the same way as pharmacists generally have supplied domestic remedies for human use. At one time these were mostly if not all prepared by the pharmacist himself but latterly, in common with the practice in human medicine, there has been a progressive replacement of locally prepared medicines by ready-packed medicines manufactured on a large scale and for the same reasons.

The pharmacist has not to any substantial extent played a complementary rôle to that of the veterinarian in supplying the medicines prescribed by the latter, and it has been the general custom for veterinary practitioners whether qualified or unqualified to supply themselves most of, if not all, the medicines needed for animals being treated by them. They may prepare the medicines themselves or obtain them ready made, just as the pharmacist does, and buy from the same wholesale and manufacturing firms as the pharmacist or from firms who supply veterinary surgeons only.

Parallel with the supply from country pharmacies there has developed in modern times a channel of supply of ready-packed veterinary remedies direct from the manufacturer to the farmer. Certain firms specialise in making and supplying animal medicines in this way. Also dealers in agricultural requirements, forage merchants and so on often engage in the sale of ready-packed preparations to farmers.

Reference must also be made to the supply of medicines for domestic animals. Such medicines are widely sold by pharmacists, " pets " shops and corn dealers. The medicines involved are mainly specialities manufactured by firms who specialise in such remedies.

The absence of effective legal control over the conditions under which veterinary products are prepared and sold has assumed a more serious aspect as the importance of maintaining a prosperous livestock industry has come increasingly to be recognised and new and more potent remedial agents with greater potentialities for harm as well as good have been added by modern research to the veterinary materia medica. Nor is the need for such control based solely upon economic considerations. The moral duty to protect animals from unnecessary suffering weighs largely with those who are concerned to see reforms introduced.

The introduction and increasing use in recent years of biological, chemotherapeutic and antibiotic substances has perhaps effected an even bigger transformation in the field of veterinary medicine than in that of human medicine. British pharmaceutical firms who are well known for their products in human medicine have been prominent in this field and have contributed largely to the successful development of newer types of products for the prevention and treatment of animal diseases.

In the absence of legislative control and in consequence of the hazards associated with their products, some manufacturers have taken steps to ensure that products which by reason of their potency or the purpose for which they are used should be administered by or on the advice of qualified veterinarians are in fact so administered, both in the interests of animal welfare and of the reputation of the product and its manufacturer, who might be faced with an action for damages if animals were injured or lost in consequence of misuse of the product. The products so affected may, on the one hand, be supplied through pharmacies either on an undertaking being given that they will be supplied only on the prescription or order of a veterinary surgeon or only to a named veterinary surgeon or, on the other hand, the products may be supplied only to a veterinary surgeon. The practice of firms in this regard varies.

The importance of some control being exercised over the supply of these substances in the absence of statutory regulation has been recognised by the Pharmaceutical Society, which has discussed the matter with the two representative bodies of the veterinary profession, the Royal College of Veterinary Surgeons and the National Veterinary Medical Association, with a view to an agreed policy being applied.

Action has also been taken to bring about statutory control of the conditions under which biological substances are manufactured.

For many years the manufacture for sale of biological and other substances for human use which cannot be tested by chemical means has been subject to rigid statutory control. While similar statutory powers have existed to deal with such substances for veterinary use they have not hitherto been used. Representations have been made in recent years by the veterinary profession and animal welfare interests with which the Pharmaceutical Society has associated itself, to urge the Government to use the powers which they possess in this connection to prevent unnecessary suffering among animals used for preparing and testing biological substances and to ensure that the substances are of proper strength and purity. The Government have agreed to do so and the necessary measures are now being worked out.

So far as the prospect of legislation to deal generally with veterinary medicines is concerned there is little to report. The Government committee conducting the enquiry into veterinary practice by unregistered persons, to which reference has already been made, recommended *inter alia* that the advertising and sale of veterinary medicines should be controlled by the Ministry of Agriculture and Fisheries, but this recommendation has not yet been implemented. The matter is, however, receiving attention by the professional bodies concerned.

Some years ago a stage was reached in developments affecting the veterinary and pharmaceutical professions which indicated that a closer official connection between the two than had hitherto existed was desirable. Since then representatives of the Pharmaceutical Society and the veterinary organisations have discussed matters of common interest and results of mutual advantage have accrued.

Reference has already been made to some of the matters which have been or are under discussion, and I should

like to conclude these remarks by mentioning what perhaps has been so far the most important result of this co-operation.

This is the preparation of a work of reference on veterinary drugs and medicines which will be called the British Veterinary Codex. It will be compiled on lines similar to those of the British Pharmaceutical Codex which the Pharmaceutical Society has published since 1907 and which has become a recognised work of reference giving descriptions and standards for drugs not included in the British Pharmacopoeia and authoritative information about their action and uses.

In view of the absence of suitable standards for veterinary drugs and the shortage of books dealing adequately with veterinary materia medica the suggestion that a veterinary counterpart of the British Pharmaceutical Codex should be prepared was taken up with enthusiasm in veterinary and pharmaceutical circles. In spite of the experience gained in many years of publication of the British Pharmaceutical Codex and the existence of the necessary editorial assistance in the staff of the Society's Scientific Publications Department, the preparation of the British Veterinary Codex presented a formidable task requiring the full and close co-operation of numerous veterinary, pharmaceutical and other experts in different branches of veterinary medicine and pharmacy over many months.

A Committee, consisting of seven veterinary surgeons and five pharmacists, entitled the British Veterinary Codex Committee, was set up in April, 1950, to assume general responsibility for the work and was followed shortly after by a number of sub-committees to deal with various aspects of the work with a total personnel of 60. Corresponding members of the main Committee were appointed in Australia, Canada, South Africa, India, the Republic of Ireland, New Zealand, Northern Ireland, Pakistan, South Africa and the United States.

The book will contain about 520 monographs together with a number of formulae and appendices and is expected to be published in 1953. We hope it will prove of value not only to the veterinarians and pharmacists of the British Commonwealth and Empire but to those of other countries as well.

### THE ASSOCIATION OF UNIVERSITIES OF THE BRITISH COMMONWEALTH

The third conference of senior university representatives from the countries of the Commonwealth will open in New Delhi on December 21st. It will discuss matters of common interest to universities in the Commonwealth. The President of the conference will be Dr. A. L. Mudaliar, Vice-Chancellor of the University of Madras, who is this year's Chairman of the Association of the Universities of the British Commonwealth. The conference will be attended by 17 visiting representatives from universities of other Commonwealth countries, in addition to the Vice-Chancellors of all Indian universities. After the conference the delegates will visit some Indian universities. At Madras a meeting will be held of the Executive Council of the Association of the Universities of the British Commonwealth.

* * * * *

### AGRICULTURE AND FOOD COMMITTEES MERGED

In conformity with the Government's policy of establishing co-operation between the food production and food supply services, the two Conservative Parliamentary Committees, the Agriculture and Food Committees, have now become one. Mr. Anthony Hurd has been elected Chairman of the new Agriculture and Food Committee and other officers elected are: Vice-chairmen, Captain James Duncan and Mr. C. M. Thornton-Kemsley; Hon. Secretaries, Mr. J. Baker White, Mr. W. R. A. Hudson, and Mr. D. G. Bullard.

## CLINICAL COMMUNICATION

## CHLOROMYCETIN* THERAPY IN AN OUTBREAK OF HARD PAD

M. B. TOOSEY, M.R.C.V.S.,
Wimborne, Dorset

*Subjects.*—Forty-two adult coursing greyhounds, over 10 months ; 27 greyhound puppies bred for coursing, under 10 months.

*History.*—July 4th. Three dogs in the training kennels were reported to be showing severe signs of illness and to be off their food. Five more dogs were found to be affected on examination after arrival.

*Symptoms.*—All eight dogs showed temperatures ranging from 103·6° to 105·9°, with marked lethargy, injected mucous membranes, and severe tonsillitis. Conjunctivitis and ocular discharge were noted.

Severe fits were reported by the owner, in the case of one of the first three patients.

*Diagnosis.*—Hard-pad disease.

*Further Histories.*—During the subsequent four weeks all 42 adult dogs developed clinical signs of the disease.

The 27 puppies, which were kept isolated as far as was practicable, also started to become affected six weeks after the original outbreak.

The symptoms shown by these later cases did not differ markedly from those of the original eight dogs.

Treatment. Because of the results obtained by the use of chloramphenicol in virus diseases in man, the drug was used in May, 1951, on six cases of hard-pad disease. Consistently good results were obtained, and for this reason the use of the drug in the outbreak under review was decided upon.

*The Original Eight Dogs.*—Chloromycetin (chloramphenicol P., D. & Co.) administered orally. Initial dose 0·5 gramme, followed by 0·25 gramme *t.d.s.* per dog for four days.

At the same visit each of the in-contact dogs was given 20 c.c. of hyperimmune hard-pad serum as a prophylactic; it was found in the light of later experience that this serum seemed to have had no beneficial effect in preventing or allaying the severity of further outbreaks of the disease.

*Treatment of Later Outbreak.*—This was the same as that for the original eight dogs, allowing for differences in dosage of chloromycetin worked out on a weight-ratio basis. In the early stage as further dogs were shown to have become in-contacts serum was administered to them, but when later these passively immunised dogs also developed the disease the use of serum was abandoned.

Course. *The Adult Dogs.*—In most cases from 8 to 24 hours after the administration of chloromycetin a marked drop in temperature and a general clinical improvement was noted. By 48 hours after initial dosage 75 per cent. of the patients showed a normal temperature. This improvement continued and at the end of three days most of the patients were eating well.

Individual dogs showed one or more of the following additional complications during the course of the disease: diarrhoea and stomatosis, emaciation, pulmonary congestion

* Chloramphenicol: Parke, Davis & Co.

and pneumonia, nervous disturbances, including fits and paralysis.

Of these adult dogs, one had not shown any appreciable rise in temperature, although she was noticed to have periodic bouts of inappetence and lethargy from as early as the second week after the outbreak. Chloromycetin was not administered to this dog until during the sixth week of the outbreak when she broke down and developed hyperkeratosis of the pads, which subsequently sloughed. After the administration of chloromycetin her recovery followed the same lines as the previous cases.

Ten per cent. of the adult dogs showed signs of relapse after a varying period of days following the cessation of administration of chloromycetin, with a rise in temperature, anorexia and general malaise and, in two cases, pneumonia. In cases of whooping cough in human medicine treated with chloromycetin relapses have also been recorded which respond well to a second course. The dogs showing relapses also recovered when treated with a second course. Streptomycin intramuscularly gave good results when combined with chloromycetin in the cases of pneumonia. The use of penicillin alone on the cases showing symptoms of relapse met with no success and was discontinued.

Kaolin and phthalylsulphathiazole were administered in cases of diarrhoea with good results.

Benerva compound was administered extensively to all cases throughout the outbreak.

No casualties attributable primarily to hard-pad disease have occurred and at the present time all the adult dogs have been perfectly healthy for the last five weeks and training will commence in three weeks' time.

*The Puppies.*—The same general plan was followed as for the adult dogs, although the younger the animal the more severe were the symptoms, suckling and newly weaned puppies suffering most. Hard pads and other complications were more common amongst this group.

One puppy died, and three more were destroyed as it was considered that the setback at so early an age would affect their future as adult coursing dogs.

The reactions of this group to the chloromycetin therapy were good, if not quite so spectacular as those of the older dogs.

All the puppies show clinical recovery, although 16 are still under observation.

November 12th. All adults and puppies clinically normal with no apparent ill effects.


### Summary

(1) The use of chloromycetin in hard-pad disease is reported.

(2) Good results have been obtained.

(3) Nervous sequelae of hard-pad disease have been minimised to a high degree in this outbreak.

(4) Further investigation into the use of this drug in hard pad and allied virus diseases would appear justified.


*Acknowledgments.*—I would take the opportunity of expressing thanks to Mr. J. Birrell, M.R.C.V.S., for his kind help and suggestions, also to the owner of the kennels for unremitting co-operation in nursing and in maintenance of records during the outbreak.

### Weekly Wisdom

*To-day nobody knows the real cost and the real price of anything. All costs and all prices are hidden under multifarious and impenetrable disguises. It is imperative that these disguises should be removed and the true state of affairs brought to light.*—Paul de Hevesy (letter in *The Times*).

## ANNOTATION

## DIAGNOSING THE TUBERCULOUS UDDER

Outstanding contributions to this subject were made by Torrance (1922, 1927, 1936). He emphasised the importance of examining the udder and supramammary lymph-nodes from behind in order to make a careful comparison between the two sides (1922). This view was again emphasised by Steward (1950) following the article of Weir and Barbour (1950). Torrance (1927, 1936) described the types of disease encountered and, perhaps most important of all, he drew attention to the typical cell groups found in tuberculous milk (1922, 1927) and their importance in the microscopic detection of tubercle bacilli. He concluded that "for the rapid elimination of the cow with a tuberculous udder from a herd, in about 80 per cent. to 90 per cent. of the cases there is no quicker method known than a capable veterinary surgeon who can . . . interpret what he sees under the microscope," a view supported by the survey carried out by Steward (1937).

Maitland (1937) compared various techniques for the microscopic examination of milk, and concluded that the following, which is similar to that of Torrance, and Matthews (1931), was the most efficient :—

(*a*) Cell-groups are most easily found in the deposit from milk which has been centrifuged for three minutes at about 2,500 r.p.m. More rapid or prolonged centrifugation is detrimental.

(*b*) Films of deposit are best prepared by pouring off all cream and fluid after centrifugation. The tube is then held inverted and, using a wire loop bent at right angles, two loopfuls are transferred to a clean new slide. This material is then spread with another glass slide as in the making of a blood film. This procedure is advantageous because cell-groups tend to collect at the edge of the smear and are thus more easily found.

(*c*) Films so prepared should be dried in air for at least 30 minutes and *lightly* fixed in a flame. They should then be defatted with a mixture of equal parts of alcohol and ether acting for 15 minutes and stained by the Ziehl-Neelsen method. By using glass slide holders standing in photographic dishes containing stain (carbol fuchsin can be heated in a stainless steel dish), large batches of smears can be stained simultaneously.

(*d*) Stained films are examined under low magnification for the presence of cell groups and, when found, these groups are examined under high magnification for the presence of acid-fast bacilli.

The adoption of the above technique raised the microscopical examination of milk samples to a high level of efficiency and, when quarter samples from 950 cows were examined in this manner, the findings were in agreement with the results of the biological examination of the milk or the *post-mortem* examinations of the cows, in all but a single instance.

It would seem that at least 90 per cent. of tuberculous cows may be detected by microscopic examination : the importance of this method hardly needs emphasis when one bears in mind the slowness of cultural and biological methods for the detection of tubercle bacilli. It has been generally assumed that the method is suitable only for the examination of milk from individual cows, and in a recent paper Maitland (1950) concludes that, used in this way, the method is "practically as sensitive a test as guinea-pig inoculation." With the assistance of Mr. R. D. Locke, M.R.C.V.S., she has extended the method to the examination of bulked milk

from groups of 10 cows. The cows on 108 farms, from which a bulk milk sample had infected guinea-pigs, were examined. Milk was examined microscopically from individual cows that were suspicious clinically and the milk from the remaining cows was bulked in groups of 10 and the bulk sample examined by the same microscopic technique as for individual cows ; if any bulk sample was positive, further examinations were carried out on the individual cows. Using these methods, 88 per cent. of the 108 farms in this series were clear of cows giving *Myco. tuberculosis* in the milk 24 to 72 hours after Mr. Locke's first visit to the farm, and the remaining 12 per cent. were cleared within six to seven weeks.

A series (number not stated) of routine bulk milk samples was examined microscopically, and by guinea-pig inoculation ; two of three positive samples detected by guinea-pig inoculation were also detected microscopically. Considering the speed and economy of microscopic examination compared with guinea-pig inoculation, Maitland concludes that the advantages of examining bulk milk samples microscopically far outweigh the disadvantages.

J. F.


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## ABSTRACTS

### Disc Protrusion in the Dog*

The work described in this study was carried out at the Royal Veterinary College, Stockholm, during the years 1949-51.

The study embraces 33 dogs, some of which showed protrusion of more than one disc so that in all 54 disc protrusions were observed.

By direct methods, X-ray examination shows that only 50 per cent. of cases can be detected. The radiological changes observed being, as calcification of the protrusion, diminished space between the surrounding vertebrae, calcification of the disc and osteophytic changes of the surrounding vertebrae.

By utilising a contrast medium it is claimed 37 out of 54 disc lesions could be detected.

After considering the various contrast media available, the author decided to use thorotrast in spite of the known objections to this substance, namely, that it is irritant, elimination is slight, there is a risk of increased intra-cranial pressure and the substance is slightly radio-active.

Under nembutal anaesthesia the contrast medium is introduced to the *cisterna magna* by intrathecal injection through the atlanto-occipital space. After the injection is made, the dog is laid on a table slanting at an angle of 30 degrees.

Protrusion of the disc alters the contour of the spinal cord as delineated by the contrast medium.

The author states that " the great complexness of the symptoms in disc protrusion in the dog renders it difficult to classify the disease into limited symptomatic groups." Posterior stiffness, paraplegia, pain when touched and slightly rigid abdominal muscles were the signs more commonly observed. The author goes on to state, " *judging from obtained results, it is most likely not an overstatement to say that most so-called rheumatism and myalgia in breeds exposed to disc protrusions are symptoms caused by such protrusions.*" The breeds referred to may be described in general terms as those with short legs and long backs, such as the Dachshund and Pekinese.

The mechanism of disc protrusion is discussed. It is thought that rupture of the *annulus fibrosus* permits protrusion of the *nucleus pulposus* under the influence of pressure exerted on the disc when the animal moves. The symptoms which follow protrusion of the disc depend on the location of the protruding disc or discs and the degree of compression of the spinal cord. Gradual stabilisation of the protrusion will prevent pressure on the cord and thus the symptoms will wane ; this may be the mechanism of spontaneous recovery. Relapses are liable to occur when the disc or discs are subjected to renewed strain. It is suggested that intermittent oedema of the herniated material may be responsible for the recurrent episodes.

Surgical intervention in the form of laminectomy was not successful. It was decided to try the method of disc fenestration ; this aims at unloading the pressure on the disc by making an opening into the disc dorso-laterally. The affected and adjacent discs could be fenestrated at the same time. Twenty-eight dogs were subjected to fenestration One died on account of the operation, one died from intercurrent disease, and one was killed two months after the operation. Twelve dogs recovered completely and 13 showed a partial recovery of locomotion.

Comparison with a number of dogs treated by rest and the administration of vitamin B appeared to show a better recovery rate in those animals treated by disc fenestration, but the author points out the comparison does not necessarily furnish final evidence since the results may be influenced by spontaneous recovery.

Twenty dogs with disc protrusions were subjected to *post-mortem* examination by section of the spine. This investigation showed that even very small protrusions may be diagnosed by thorotrast myelography provided the contrast medium reaches the site of the protrusion ; this may be prevented by inflammatory swelling in the vertebral canal and haemorrhages in the subarachnoid space.

G. F. B.

* * * * *

### New Chemotherapeutic Agent to Arrest Bleeding*

The agent employed was Koagamin—a proprietary product comprising an aqueous solution of oxalic and malonic acids.

The author reports the use of this product on 455 animals in conjunction with surgery, and also in the treatment of traumatic injuries and of haemorrhages associated with infectious disease. The drug was given intramuscularly in doses of 1 to 2 c.c., followed where necessary by a subsequent dose given intravenously (often dissolved in glucose and saline solution). No ill effects were observed. The results were considered to be good but, with one exception, no control figures are given for cases where Koagamin was not used. The exception is in connection with the operation for pyometra (13 cases) where it is considered that the use of this preparation may have contributed to a fall in the death-rate from 50 per cent. to 35-40 per cent.

S. W. D.

* **On Disc Protrusion** (*Enchondrosis tubervertebralis*) **in the Dog.** Olsen, S. E. (1951.) *Act. Orthop. Scand.* Supplement N. VIII. pp. 76. (Figs. 40.)

* **The Use of a New Chemotherapeutic Agent to Arrest Bleeding in Animals.** Klausman, B. S. (1951.) *Vet. med*. **46**. 282-283.

## REVIEWS

**[Ethylene and Propylene Glycols.** By John Rae, M.P.S. *Pharmaceutical Press.* 2s. 6d.]

This collection of articles published over a number of years is concerned with possible applications of both the synthetic dihydric alcohols, ethylene and propylene glycols, the greater importance of the latter being stressed. Some indications for their use as solvents and preservatives of vegetable drugs and colouring agents are illustrated by experimental data, and a case is made for the use of propylene glycol in the extraction of such important compounds as anthraquinone derivatives and morphine.

Evidence is given that, as a preservative, propylene glycol is equal, and in some cases superior, to either of the related solvents ethyl alcohol and glycerine. It is also shown that moulds and enzymes are markedly inhibited but that penicillin is rapidly destroyed by these glycols. On pages 25 and 26 there would appear to be a discrepancy between the text and the summary concerning the relative efficacy of alcohol and propylene glycol as solvents for ginger, but apart from this the experimental findings are clearly set out in tabular form and, in most cases, a summary is given at the end of each article.

It is a pity that dietetic and pharmacological experimental evidence is not also available, an omission to which attention is drawn by the reference to toxicity in the introductory paragraphs.

* * * * *

### Pony Club Annual, 1952

The British Horse Society are to be congratulated on their production of the Pony Club Annual for 1952. Only two years have passed since the first edition was published, and it is hoped that this book will soon become an annual feature.

The great diversity of subjects dealt with will interest all, be they young or old, and it will not be the fault of the numerous authors if something both practical and useful is not learned.

This book is well set up on good paper and amply illustrated. It is, perhaps, a little unfortunate that some of the excellent pictures and illustrations are not included in their appropriate text.

The Annual can be recommended to persons of all ages who have an interest in and love of horses.

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*Also received*—"Nature of Disease Institute: Third Annual Report." By J. E. R. McDonagh, F.R.C.S. Published (September 14th, 1951) by William Heinemann Medical Books, Ltd., 99, Great Russell Street, London, W.C.1, price 21s. net.

## QUESTIONS IN PARLIAMENT

### Transit of Horses (Inspection)

Lieut.-Colonel J. C. Lockwood (November 15th) asked the Minister of Agriculture how many of his officers have been appointed to carry out inspection under the Transit of Horses Order, 1951; and what has been the result of their investigations to date.

Sir T. Dugdale: No officers have been specially appointed to carry out inspections under the Transit of Horses Order, 1951, as that work forms a part of the normal duties of the veterinary staff of the Department. Any infringements of the Order would be brought to the notice of the police, who, with local authorities, have the duty of enforcing it. There is one case of difficulty which is being dealt with, but I do not know of any other failure to observe the provisions of the Order.

### Electric Milking Apparatus

Mr. Peter Freeman (November 15th) asked the Minister of Agriculture if he will introduce legislation to prevent accidents arising from the faulty use of electric milking apparatus; how many cases have been reported to him; and what steps are being taken to prevent any recurrence.

Sir T. Dugdale: I understand that a few cases of cows having been electrocuted in byres in recent years have been reported. Under Regulations, electricity boards require any installation to comply with certain safety standards, and I am not satisfied that further safety provisions are needed.

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### Ministry of Food: Condemned Carcases

Mr. Peter Freeman (November 14th) asked the Minister of Food how many cattle and cows were killed for food in 1950; how many were inspected; how many were totally condemned as unfit for human consumption, and for what reason; and how many carcases there were of which some part or organ was condemned and the percentage in each case.

Major Lloyd George: The total numbers slaughtered for sale for human food in Great Britain in 1950 and the numbers wholly condemned after slaughter were as follow:—

| | *Number slaughtered* | *Number of carcases wholly condemned* |
|---|---|---|
| Cattle (other than cows) ... ... | 1,580,409 | 11,657 |
| Cows ... ... ... ... ... | 634,892 | 34,284 |

All animals slaughtered on behalf of my Department may be inspected by the meat inspectors of the local authorities. Where animals are accepted for slaughter as casualties or on the basis of owner's risk it is a condition of purchase that the carcase shall be examined by the meat inspector as the payment to the producer is based on the weight of the carcase, or part of the carcase, passed fit for human consumption. 117,145 cows and 189,884 other cattle were accepted under the latter arrangements and of these 20,556 cows and 6,110 other cattle were totally condemned and are included in the totals given above.

Records are not yet available from all local authorities of the number of carcases of which parts only were condemned but we have records of the total weight of all condemned meat, including parts of carcases, and the percentage which this bears to the total weight of meat produced on slaughter during this period was as follows:

| | |
|---|---|
| Cattle (other than cows) ... ... ... ... | 0·74 per cent. |
| Cows ... ... ... ... ... ... ... | 5·36 „ „ |

Records are not yet available from all the local authorities of the total number of carcases examined by their meat inspectors or of the reasons for condemnation.

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### Coal Mines (Horses and Ponies)

Mr. Peter Freeman asked the Minister of Fuel and Power the number of horses used below ground in coal mines at the end of June, 1951; and the number used below ground in the Durham and Northern (N. and C.) Divisions, as well as in the other divisions, at the same date.

Mr. Geoffrey Lloyd: The number of horses used below ground in coal mines at June 30th, 1951, was as follows:—

| *Division* | |
|---|---|
| Scottish ... ... ... ... ... ... | 139 |
| Northern (N. and C.)* ... ... ... | 2,427 |
| Durham ... ... ... ... ... | 6,870 |
| North Eastern ... ... ... ... ... | 2,582 |
| North Western ... ... ... ... ... | 59 |
| East Midlands ... ... ... ... ... | 1,333 |
| West Midlands ... ... ... ... ... | 776 |
| South Western ... ... ... ... ... | 3,154 |
| South Eastern ... ... ... ... ... | — |
| Great Britain ... ... ... ... ... | 17,340 |

Mr. Peter Freeman asked the Minister of Fuel and Power the average annual rate of reduction in the number of horses and ponies used in coal mines in each of the 10-year periods from 1920 to 1930, 1930 to 1940 and from 1940 to 1950; and the reasons for the reduction rate in the respective periods.

Mr. Geoffrey Lloyd: The average annual rates of reduction in the number of horses and ponies used in coal mines in the 10-year periods ended 1930, 1940 and 1950 were 1,810, 1,880, and 1,250 respectively. These reductions reflect the increasing mechanisation of the mines.

# NOTES AND NEWS

### Diary of Events

Nov. 27th.—Unveiling of War Memorial to former Students of the Royal (Dick) Veterinary College, in the War Memorial Library, 3 p.m.

Nov. 28th.—Whole-day meeting of the Technical Development Committee, N.V.M.A., 10.30 a.m.

Nov. 30th.—North Wales Division, N.V.M.A., Dinner-Dance at Llandudno (St. George's Hotel), 7.30 p.m.

Nov. 30th.—Meeting of the Royal Counties Division, N.V.M.A., at Abingdon (Guildhall), 2.30 p.m.

Dec. 6th.—Meeting of the Central Veterinary Society at the Royal Veterinary College, N.W.1, 6 p.m.

Dec. 10th.—Meeting of the Southern Counties Division, N.V.M.A., at Southampton (Polygon Hotel), 6 p.m.

Dec. 12th.—Meetings of the Editorial Committee, N.V.M.A., at 36, Gordon Square, W.C.1: Executive, 2 p.m.; Main Committee, 3.30 p.m.

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### R.C.V.S. EXAMINATIONS

*Thursday, November 29th.*—M.R.C.V.S. Written Examinations.
*Friday, November 30th.*—Ditto

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## Emergency Assistance Scheme for Single-handed Veterinary Surgeons in Practice

The Home Appointments Committee of the Association has for some time been considering ways and means of giving more help to those requiring qualified assistants in an emergency.

After consulting Divisions, it was found that many of them were willing to co-operate with certain provisos: (a) that the assistant being sent to help should not have to go too far away; (b) that he should go for only a few days; (c) that the principal agreeing to help in this scheme should not be expected to do so if, at the time of asking, it was out of the question for him.

The Council of the Association has accordingly approved the possibility of working such an arrangement from headquarters and is notifying Divisions of this. Members interested are now asked to send their names either to the Hon. Secretary of their local N.V.M.A. Division, or direct to the General Secretary, so that they can be added to this proposed register.

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## Calfhood Vaccination Scheme

In the September 15th, 1951, issue of *The Veterinary Record* (page 600) we published a Ministry of Agriculture Press notice concerning the above scheme under the heading, "Vaccination against contagious abortion with S.19." The object of this was to give greater publicity to the value of calfhood vaccinations.

Owing to the reduction which was made early this year in the number of veterinary inspections in non-designated herds in England and Wales, the opportunities for carrying out routine calf vaccinations in such herds have decreased.

The Veterinary State Medicine Committee of the Association felt somewhat concerned about the possible reduction in the number of calves vaccinated, and as a result the above-mentioned publicity was given to the scheme. The Committee and Council of the Association urge members in practice to take every opportunity to encourage their clients to take advantage of and continue to participate in the scheme, particularly those owning non-designated herds. It is hoped that all members will co-operate fully in this respect.

## Control of Distribution of Veterinary Products

At a recent meeting of representatives of the Royal College of Veterinary Surgeons, the National Veterinary Medical Association, the British Medical Association and the Pharmaceutical Society of Great Britain, called to discuss the control of veterinary biological and allied products, it was unanimously agreed that such control need not include the following products which would therefore be made freely available to the public:

Blackleg vaccine, aggressin and serum.

Preparations of synthetic oestrogens used for the chemical caponisation of poultry.

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### PERSONAL

#### Appointments in the Colonial Veterinary Service

The Colonial Office announces the following promotions, transfers and appointments in the Colonial Veterinary Service:—

Messrs R. A. Hammond, O.B.E., M.R.C.V.S., Deputy Director of Veterinary Services, Kenya, as Director of Veterinary Services, Kenya; E. C. S. Dawe, M.R.C.V.S., Director of Veterinary Services, Bechuanaland, as Deputy Director of Veterinary Services, Tanganyika; H. W. C. Newlands, M.R.C.V.S., Senior Veterinary Officer, Tanganyika, as Regional Assistant Director of Veterinary Services, Tanganyika; W. G. G. Pevic, M.R.C.V.S., Senior Veterinary Officer, Tanganyika, as Regional Assistant Director of Veterinary Services, Tanganyika; J. E. R. Roe, M.R.C.V.S., Senior Veterinary Officer, Uganda, as Director of Veterinary Services, Bechuanaland; P. J. Sheehy, M.R.C.V.S., Veterinary Officer, Northern Rhodesia, as Veterinary Officer, Bechuanaland, and L. S. H. Walshe, M.R.C.V.S., Veterinary Officer, Sierra Leone, as Senior Veterinary Officer, Sierra Leone. Mr. J. A. Hammond, M.R.C.V.S., is appointed Veterinary Officer, Tanganyika.

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*Births.*—Evans.—On October 31st, 1951, at Farnham, Surrey, to Joan (*née* Barrett), wife of L. A. Evans, M.R.C.V.S., a daughter.

Kirkwood.—On November 8th, 1951, to Patricia (*née* Brown) and Andrew Kirkwood, M.R.C.V.S., a brother for Juliet.

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### R.C.V.S. OBITUARY

Edwards, John Augustus, O.B.E., Captain late R.A.V.C., The Cedars, Ringwood, Hants. Graduated London, December 22nd, 1914. Died November 11th, 1951.

Ferguson, Julius Delmerge, 12, Arcadia Road, Umtali, Southern Rhodesia. Graduated Dublin, December 11th, 1908. Died July 24th, 1951.

* "Mr. Ferguson qualified at Dublin in December, 1908, after having saved up enough capital in the United States, where he was a 'cowpuncher' for a number of years. A man of immense stature with a very sincere love of animals, he had one great handicap and that was deafness. I believe he had both his ear drums broken while bronco-busting. He died in his home in Umtali, Southern Rhodesia, on July 24th, 1951, leaving a widow, daughter and son."

* Extracted from a letter received from Mr. J. B. Condy, M.R.C.V.S., Veterinary Department, Francistown, Bechuanaland Protectorate.

Mr. J. A. Edwards, O.B.E., M.R.C.V.S.

On qualifying from the London College in 1914, Mr. John Augustus Edwards, whose death we record above with much regret, was commissioned into the R.A.V.C., in which he served in France, being mentioned in despatches and obtaining the O.B.E. He then practised in Westmorland and later in Lincolnshire, going to Ringwood in 1936. He was a typical example of a hard-working single-handed veterinary surgeon who lived only for his work and always put his clients and their animals before his own ease or comfort.

When a sudden stroke 18 months ago prevented him from visiting, he continued to take an active interest in the practice from his bed, by the side of which to within a month of his death the telephone still stood.

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### UNIVERSITY OF LONDON B.Sc. (VETERINARY SCIENCE) EXAMINATION

#### Pass List for October, 1951

Following is the Pass List for the above examination, October, 1951:—Early, James Lincoln; Faull, William Baines; Hawksley, Michael Bruce; Holcombe, Robert Brian; Howell, Dennis George;

Jull, David John; Rees, William Howard Guest and Sainsbury, David William Bateman.

The following have passed in Part II only:—Ahmad, Alauddin; Cripps, Jack Harry Harrison and Jenner, Francis Harold.

All are Internal Students of the Royal Veterinary College.

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## ROYAL COLLEGE OF VETERINARY SURGEONS

### Pharmacology, etc., Examination

List of Successful Candidates

(*Continued from page 727*)

Dublin:

Cullen, R. G.
Hernon, P. C.
Houlihan, T. V.
Kelly, E. J.
Magee, B. F.
Marsh, J. H.
McElroy, Robert
O'Keeffe, D. J.

### Animal Management Examination

List of Successful Candidates

(*November*, 1951)

Dublin:

Barry, K. T.
Daly, Edward
Gahan, M. A. B.
Guven, M. N.
Henehan, M. D.
Jameson, J. G.
McElroy, Robert
O'Connor, T. J.
Walsh, M. C.

Glasgow:

Torrance, D. S.
Watson, J. D.

Edinburgh:

Black, Hugh
Black, Michael
Cock, E. V.
Cuthbertson, J. L. H.
Forsyth, D. W.
Grant, Forbes
Hayes, Hilton
Humphreys, Medwyn
McIntosh, S. A.
McKay, A. F.
Nelson, A. M. R.
Sharratt, R. K.
Watson, A. R. A.

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## LEGAL NOTES

### Dispute Over Treatment Given to Heifer

Judge Rejects Allegation of Negligence Against Veterinarian Receiving Mutual Defence Society Support

A farmer's dispute over the treatment given to a heifer's foot by a veterinary surgeon and his refusal to pay the bill was heard by Judge Neal at Rochester County Court recently, when Mr. Derek G. Lewis, veterinary surgeon, of High Street, Rochester, was plaintiff in an action against E. Smith & Sons, farmers at Allhallows. Messrs. Smith counter-claimed for £35, the value of the beast less the sum of £4 15s. which they received for the animal when slaughtered. They alleged negligence, and that they had not authorised the removal of part of the animal's foot, an operation which had resulted, they said, in the heifer's subsequent inability to walk and feed properly so that it had to be slaughtered about six months after the operation.

Mr. Lewis denied negligence.

Mr. Peter Boydell was counsel for the plaintiff, whose case was in the care of the National Veterinary Benevolent and Mutual Defence Society, and Mr. Brian Galkin was counsel for defendants.

Plaintiff stated that he first examined the animal's foot on the Binney marshes on May 22nd, 1950, and found a tear extending around one of the two digits, 3 inches above the hoof, with extensive gangrene. He thought that the heifer had been caught in barbed wire. "I told Mr. J. B. Smith (the father) that in my opinion it was either a question of removing the affected digit or slaughtering the animal, and he said he would discuss it with the two sons. The operation was agreed to. The father was not present when I removed the digit two days later. I was assisted by the son. I called two days after and re-dressed the wound, and again on May 30th, when I removed splints and examined the foot in the presence of one of the sons. I was pleased with it. I re-dressed it and gave the son a packet of M & B powder, and told him to change the dressing. I called again on June 2nd and the wound was improving. I said I would try to get there on June 5th, but if anything happened to give me a ring. I was very busy that day and decided the heifer could wait till the next day, but that night I fell ill and from then until five days later I was in bed, with my assistant carrying on. We did not consider the heifer a priority case, and we had had no calls from the Smiths."

Plaintiff then described how on June 19th he again examined the animal. "The foot was looking much healthier, I was pleased with it and I repacked it," he said.

Counsel for Messrs. Smith said that what had been said at the time of the first examination was that the horny part of the hoof should be pared off. "We say this animal went through an operation more serious than necessary, that the veterinary surgeon did not examine it for 17 days, and after that it was touch and go whether it was saved or not."

"Do you think it right that an animal with a temperature should be left for that long only eight days after an operation?" Mr. Brian Galkin asked the plaintiff. "I don't think it should," replied plaintiff, "but the onus was on Smith to 'phone. I never thought the animal was in serious danger. I told them to ring and I relied on that."

Defendants were Mr. John R. Smith, his son Alan Smith and nephew Hubert Smith. Mr. Smith, senr., in evidence said that plaintiff had said he would take off the hoof. "I thought that correct. I have done sheep that way, by paring the hoof to get at the seat of the trouble. He said nothing of slaughtering. I was not there at the time he dealt with it and when I saw the heifer that night I was disgusted to see it lying there in such pain. If I had been there I wouldn't have let him do it."

Judge Neal: Do you consider you know better than a veterinary surgeon?—I have had 57 years' experience.

His Honour: If you call a veterinary surgeon don't you take his advice?—Certainly, that's what he was called for, but he didn't do what he said he would do.

Mr. Brian Galkin said that there was evidently dispute whether it was a tear or condition similar to foot rot. It was clear that the Smiths expected neither slaughtering nor the operation, but thought the veterinary surgeon was going to pare the hoof. While the foot was eventually cured to a certain extent the animal could not walk and graze, so had to be slaughtered.

His Honour found that plaintiff was not guilty of any sort of negligence and accepted his opinion that the operation was necessary. He was quite sure, he said, that it was not the claim for £4 17s. 6d. which had brought him to court, but the allegation of negligence.

His Honour said that he could not see that the plaintiff had done wrong or that the heifer suffered from his not turning up in his unfortunate illness, for he had left dressing materials with the request to be telephoned if needed. The cow had recovered as far as possible and had been slaughtered because it could not get enough nourishment. The obvious thing to have done, he thought, was to see whether the animal could not be kept in a byre and fed up.

"I have heard nothing in this case to show the plaintiff was not highly competent and reliable," said Judge Neal, and gave judgment for the plaintiff with costs.

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## FOOT-AND-MOUTH DISEASE

### Outbreaks in East and Midlands

On November 15th the Ministry of Agriculture reported that outbreaks of foot-and-mouth disease had been confirmed in the vicinity of Scarborough, Hull, Norwich, Lowestoft and Colchester, adding that inasmuch as all these places are near the coast and since foot-and-mouth disease is at present very prevalent on the continent of Europe, it is thought that infection may have been introduced from that source. "If so, we may have further cases to deal with. Farmers are therefore warned to keep a close watch on the health of their stock and report to the police immediately should anything occur to give rise to a suspicion that foot-and-mouth disease may be present."

Unfortunately this fear speedily was proved justified, and on Monday last the Ministry stated that during the past few days more than a dozen outbreaks of foot-and-mouth disease, involving the slaughter of about 1,750 animals, had been confirmed in Essex, East Suffolk, Norfolk, Nottinghamshire and the East and West Ridings of Yorkshire. As a precautionary measure, restrictions had been imposed on the movements of stock and on the holding of markets in a large portion of eastern England.

An order had been made applying the provisions of the Foot-and-Mouth Disease (Controls Areas Restrictions) General Order, 1938, to those parts of the counties of Sussex, Kent, Essex, East and West Suffolk, Norfolk, Cambridge, Huntingdon, the Isle of Ely, the Soke of Peterborough, Lincoln, Nottingham, the East Riding and the south-eastern portions of the West Riding of Yorkshire which are not within an affected area. The order controls the movement of animals into, within and out of the area and prescribes that no market or sale of animals shall be held in the area unless it is restricted to fatstock for immediate slaughter and is licensed by the local authority.

On the following day outbreaks of foot-and-mouth disease were confirmed among dairy cattle at Tilton-on-the Hill, Leicestershire, and Writtle, Chelmsford, Essex, and further outbreaks in Lincolnshire, Norfolk, Suffolk and Yorkshire (East and West Ridings).

The administrative counties of London and Middlesex have since been added to the controlled area.

## ADDRESSES OF DISEASE-INFECTED PREMISES

LIST SHOWING THE OUTBREAKS OF NOTIFIABLE DISEASES (OTHER THAN TUBERCULOSIS) CONFIRMED ON THE UNDERMENTIONED DATES

Together With

Addresses of premises on which disease occurred and, in capital letters, the name of the Local Authority concerned.

ANTHRAX

November 9th, 1951.—Barnfield Farm, Charing, Ashford, Kent (KENT); The Grange Farm, Baddington, Nantwich, Cheshire (CHESHIRE); Mables Farm, Draycot, Chippenham, Wiltshire (WILTSHIRE).

November 15th, 1951.—Moorcroft Farm, Leymoor Road, Golcar, Huddersfield, Yorkshire (YORKSHIRE, WEST RIDING).

FOOT-AND-MOUTH DISEASE

November 14th, 1951.—St. Osyth Priory Farm, St. Osyth, Clacton-on-Sea, Essex (ESSEX).

November 15th, 1951.—Manor Farm, Little Melton, Norwich, Norfolk (NORFOLK); Church Farm, Ganton, Scarborough, Yorks (YORKSHIRE, EAST RIDING); Waveney Grange, Somerleyton, Lowestoft, Suffolk (EAST SUFFOLK).

November 16th, 1951.—Poplar Farm, Thurgarton, Nottingham (NOTTINGHAMSHIRE); No. 5, Buscreek, Southwold (EAST SUFFOLK); Lakes Farm, Tacolneston, Norwich (NORFOLK).

FOWL PEST

November 9th, 1951.—Copeland Farm and Hall Nook Farm, Bretherton; Leal Cottage, Dob Lane, Little Hoole, Preston, Lancs (LANCASHIRE).

November 10th, 1951.—Brook Mount, Lytham Road, Warton; Walmer Grove Farm, Longton; Heatherfield, Ross Lane, and Moss View Farm, Moss Lane, Little Hoole, Preston, Lancs (LANCASHIRE); Brook Poultry Farm, Buttons Green, Cockfield, Bury St. Edmunds, Suffolk (WEST SUFFOLK).

November 11th, 1951.—Refuge Farm and Lytham Road, Freckleton, Preston, Lancs (LANCASHIRE).

November 12th, 1951.—Meadowcroft, Knoll Lane, Little Hoole, Preston, Lancs (LANCASHIRE).

November 14th, 1951.—All at Preston, Lancs (LANCASHIRE): Tithe Barn Meadow, Hutton; Patterdale Poultry Farm, Much Hoole; Allandale, Shirley Lane, Longton; Mosses Farm, South Road, and Weavers Fold, Bretherton.

November 15th, 1951.—All at Preston, Lancs (LANCASHIRE): Homesley, Hesketh Lane, Tarleton; School House, Old Road, Hoole; Moorley Cottages, Bretherton; Parklands Poultry Farm, Liverpool Road, Hutton; Lane House Farm, Much Hoole.

SHEEP SCAB

November 13th, 1951.—The Hill Farm, Clifton-on-Teme, Worcester (WORCESTERSHIRE).

SWINE FEVER

November 9th, 1951.—Nightlayer Drove, Chatteris, Cambs (ISLE OF ELY); Top Yard, Spring Gardens, Queens Street, Louth, Lincs (LINCOLN PARTS OF LINDSEY); The Laurels, Aslackby, Sleaford, Lincs (LINCOLN PARTS OF KESTEVEN).

November 10th, 1951.—Robin Hood Allotments, Boston, Lincs (LINCOLN PARTS OF HOLLAND); Farm Hill Cottages, Coggeshall Road, Kelvedon, Essex (ESSEX).

November 12th, 1951.—Daintree Farm, Upper Road, March, Cambs (Carcase at: Slaughterhouse, Burrows Road, March, Cambs) (ISLE OF ELY); Oak Villa Farm, Lower Green, Astley, Manchester (LANCASHIRE); Furn House, Fleet Fen, Gedney Hill, Spalding, (LINCOLN PARTS OF HOLLAND); Steels Yard, Cluit Lane, and High Street, Navenby, and Frout Street, Dunston, Lincoln (LINCOLN PARTS OF KESTEVEN); Buck Farm, Stanfield, Dereham, Norfolk (NORFOLK); Coldbath Farm, 333, Yardley Wood Road, Birmingham, 14 (CITY OF BIRMINGHAM); Cottage Holding, Dunswell, Hull, Yorkshire (Carcase at: Ministry of Food Slaughterhouse, Gt. Thornton Street, Hull, Yorkshire) (YORKSHIRE, EAST RIDING); Gore Farm, Upchurch, Sittingbourne, Kent (KENT).

November 14th, 1951.—The Garage, Offord Cluny, near Huntingdon (HUNTINGDONSHIRE); The Orchard, near the Station, Warboys, Huntingdon (HUNTINGDONSHIRE); All Saints' School, Tedmarton Road, Bloxham, Banbury, Oxon (Carcase at: Ministry of Food Slaughterhouse, Swan Close, Banbury, Oxon) (OXFORDSHIRE); Dairy Farm, Great Packington, Meriden, Coventry, Warwickshire (Carcase at: Public Abattoir, Coventry, Warwickshire) (WARWICKSHIRE).

November 15th, 1951.—Jesmondene, 28, Station Road, Alford, Lincs (LINCOLN PARTS OF LINDSEY); Amazondead, Silverburn, Penicuik, Midlothian (MIDLOTHIAN); Southages Land Piggeries, Rastrick, Brighouse, Yorkshire (YORKSHIRE, WEST RIDING); Top Croft, Stradbroke, Diss, Norfolk (SUFFOLK EAST); Sandy Lane, Old Woodhall, Woodhall Spa, Lincs (LINCOLN PARTS OF LINDSEY); Cherry Valley Farm, Rothwell, Lincoln (LINCOLN PARTS OF LINDSEY).

## THE WORK OF F.A.O., 1950-51

In his annual report, "The Work of F.A.O., 1950-51," Director-General Norris E. Dodd surveys the field of activities of the Food and Agriculture Organisation of the United Nations, region by region and country by country, describing the work being done to raise levels of nutrition and standards of living and to achieve increased supplies of food and forestry products. The report was to be reviewed by the sixth session of the F.A.O. conference on November 19th in Rome. He says ". . . nations seem at last to be preparing for a genuinely large-scale war against want." It is noted from the report that by September last 213 scientific and professional workers of 32 nationalities were working on 100 projects in 35 countries. These men work out practical methods on the spot for increasing production, improving distribution, enlarging research and experimental work, reorganising governmental agencies, and, in general, get people more acquainted with better ways of doing things. F.A.O. has been engaged on a campaign to control foot-and-mouth disease. On this the report says: "Operated on a regional basis, it will involve such activities as visits to countries by F.A.O. veterinarians and special consultants, regional meetings, exchange of information, and co-ordination of control plans; assistance in training personnel, and in conducting schools for vaccine production, diagnosis, and field control. The regional programmes will be co-ordinated through an established centre in Great Britain which has been offered as an international research laboratory."

## CORRESPONDENCE

***The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.***

### HUSK

Sir,—If the report of the discussion of the paper "Further Notes on Husk" by the Sussex Division of the N.V.M.A., published in *The Veterinary Record* of November 10th, is correct, then I am distressed not only that my old friend Dr. E. L. Taylor should misquote me so grievously, but also that he should see fit to cast even mild ridicule upon his own misquotation. I am surprised, also, that after so many years he has not yet grasped the meaning of my assertion that dead worms in the air passages might prove even more dangerous than live, active specimens.

The reason given by me was that before their death, whether this resulted from the demise of the host or the intratracheal injection of agents (cf. chenopodium in oil) which, although undesirable in many respects, actually killed the worms, the parasites tended to congregate, if the invasion were a heavy one, into dense clusters resembling tightly packed masses of cotton-wool which might completely occlude even large bronchi, a fact of which I feel sure Dr. Taylor must be aware.

My objection to intratracheal injections is based on the theory that if such injections do not kill worms they represent waste of time and money. If they *are* capable of killing worms they are liable to kill the patient also, by causing mechanical obstruction of the air passages by solid masses of dead worms.

When our parasitologists succeed in producing a non-irritant capable of killing filaria, it may be found that the above theory is not altogether ridiculous.

As regards my alleged statement, misquoted by the essayist, that "dead worms produced worse toxins than live ones," I was quite unaware that dead worms produced toxins of any importance whatsoever. In any case, I doubt that there will be sufficient interval between the respective deaths of parasite and host for these "worse toxins" to make their appearance once the "effective" parasiticide has been discovered and administered.—Yours faithfully,

R. H. SMYTHE, 45, St. James's Street, Nottingham. *November 10th*, 1951.

. . . . . .

Sir,—Soon after graduation, the fact that husk can be of major importance in adult cattle, together with the contra-indication of intratracheal injection in the presence of pneumonic symptoms, was very forcibly brought to my notice in a herd of Guernseys.

In the years that have followed it has been rare in my practice not to encounter the condition in the "husk years" in both bulling and in-calf heifers and dairy herds of all breeds. In one Jersey herd, before advice was sought, the mortality rate was high.

In my personal experience I have found that husk in adult cattle is not so amenable to treatment as in young stock, and despite

the criticisms levelled against it I still give intratracheal injection, but not if pneumonia is present.

As a drench we use "Antifilarine."

Whilst the ploughing-up of old pastures may have contributed to disease control, I rather think that this does not extend to parasitic diseases, and this may be an explanation why infection is picked up from new leys.

In view of the prevalence of husk this year the contributions published in your issue of November 10th are as interesting as they are opportune.—Yours faithfully, J. F. D. Tutt, Rothiemurchus, Winchester. *November 12th*, 1951.

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## AMPHOTROPIN IN THE TREATMENT OF HARD PAD

Sir,—The following statistics, taken over the period January, 1951—October, 1951, include only typical cases of the disease, none of which was showing any signs of encephalitis at the first visit.

The cases dealt with here had either

(1) A temperature of approximately 103° F., purulent conjunctivitis, persistent diarrhoea, and frequently a cough.

or

(2) A temperature of 102° to 106° F., purulent conjunctivitis, accompanied by bronchial pneumonia.

During this period, the number of such cases treated was 150, of which 110 recovered, giving a total recovery rate of 73 per cent.

Of these 150 cases, it is possible to take two essentially different groups, 111 in the first group and 39 in the second.

The 111 cases were treated with four intravenous injections of amphotropin on alternate days and all recovered, but later a relapse in certain cases with the development of encephalitis gave a final recovery rate of 80 per cent.

Of the smaller group of 39 there was not a complete recovery with amphotropin injections, and immediate further treatment was necessary. In these cases there was a recovery rate of only 30 per cent.

The purpose of these figures in twofold. The first is to make clear the success of the amphotropin treatment, and that it is not "merely useless," while the second is to lead others to give their experiences over a similar period of the above, or other methods. A lengthy period is necessary for a variety of reasons, among which the variability in the nature of the epidemic from time to time is not unimportant.—Yours faithfully, Joan M. Milnes, M.R.C.V.S., 83, Birkby Lodge Road, Huddersfield. *November 14th*, 1951.

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## CURARIZING DRUGS

Sir,—Mr. A. T. Cowie (*Vet. Rec.* **63.** 716) raises important issues in questioning Mrs. Sheppard's advocacy of "Myanesin" for use in veterinary practice. The whole question is integral with that of the legitimacy of the issue of "Anavenol" for use on horses (and of Anavenol-"K" for use in cattle), which is the thesis of a letter recently drafted by Mr. T. N. Gold and myself, in continuity with previous correspondence, and for publication as we hope by you at an early date.

Meanwhile, as Mr. Cowie invites comment, may I submit one or two observations specific to the drug "Myanesin?" And may I first correct an important mis-statement in Mrs. Sheppard's paper (*Vet. Rec.* **63.** 685). "Myanesin" does *not*, as she avers, "resemble curare in action by producing a block at the myoneural junction." Unlike curare, "Myanesin" acts *centrally* on the C.N.S., it is in fact specifically declared to have no curare-like action on the myoneural junctions (Berger & Bradley, 1946-47; Berger, 1947). Its mode of action has been assumed to be one of central depression; but this assumption is quite arbitrary and there are certain reasons for questioning it (Longley, 1951).

Two other matters arising in Mrs. Sheppard's paper require notice: (1) She refers to the thrombosis which has prejudiced the medical profession against the drug, and says that she has not experienced this difficulty. In laboratory rabbits I have found thrombosis to be the rule, generally with a sequel of dry necrosis, sloughing and permanent deformation of the ears. (This thrombogenesis is ascribable to the solvent.) (2) Secondly Mrs. Sheppard approved and confirmed suggestions made at the meeting that the agent might be used alone, both for the control of convulsions and diagnostically for abdominal palpation.

In a pharmacological study of "Myanesin" in rabbits, which is for future publication, I have shown, with electrocardiographic records in evidence, that this agent used alone (as opposed to its use in conjunction with the anaesthetics) has powerful cardio-inhibitory properties which in full dosage can cause abrupt, indeed almost instant, fatal syncope. Where the animal survives a common sequel is cardiac dysrhythmia, notably a bigeminal rhythm and extra-systoles (*op. cit.*). Similar incipient effects are on record in man, notably vaso-vagal collapse (fainting), on the few occasions that the drug has had use alone for neurological purposes. In human anaesthetic practice this effect has (quite fortuitously) escaped notice by reason of the fortunate concomitant use of the general anaesthetics, which completely block the effect (Longley, *op. cit.*). This important and very dangerous property of this drug is unaccountably overlooked in the introductory papers and laboratory assay of Berger & Bradley (*op. cit.*), although they worked on rabbits. To what extent the cat (and dog) are vulnerable to this action I cannot say; it is none-the-less important that the risk should be recognised.

Together with these cardiac effects there appears to be a considerable fall in blood pressure, evidenced by total collapse of the peripheral veins. In these circumstances I have observed that by raising or lowering the animal's head the bradycardia is aggravated or ameliorated respectively, exactly as in the fainting syndrome in man. This has led me to wonder to what extent the appearance of "narcosis" and unconsciousness may be rather of the nature of fainting, *viz.*, unconsciousness mediated by cerebral hypoxia, consequent on low blood pressure and slowing of the heart, than a true anaesthesia. Support seems to be lent to this concept by the recognition in human practice of a fatal post-anaesthetic nephritis of a hypoxic character, which is on record where "Myanesin" has been employed.

May I point out to Mr. Cowie that Mallinson's assertion (1947) that "Myanesin" "has a narcotic effect in animals in addition to its paralysing action" is made without proof. Indeed Mallinson is merely and uncritically quoting Berger & Bradley (*op. cit.*) to this effect. No evidence is available in the literature of human subjective experience, with loss of consciousness, with the use of "Myanesin" alone. The only practicable alternative method of assay of the effects on consciousness in animals is electroencephalography, to which no reference is anywhere made in their reports. How then can these or any other observers pretend to recognise an alleged narcosis that must be superimposed on, and which for all the evidence adduced may be completely spurious and simulated by, the prevailing general paralysis? In my submission such assertions are in the circumstances scientifically unfounded and untenable.

Apart from the obscurity of the case in this sense, however, with its humane and ethical implications; and apart from the several deleterious side effects of this drug, which would alone suffice to dissuade from its adoption, I am curious to know why it ever should be adopted? Perfectly satisfactory relaxation is attainable in the small animals by legitimate straight anaesthesia of adequate depth. And the use of "Myanesin" is in my view an expensive, unwarrantable, unnecessary and highly undesirable complication of technique.—Yours faithfully, E. O. Longley.

### References


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## TUBERCULOSIS—BOVINE/HUMAN RELATIONSHIP

Sir,—In a series of letters in your columns, Mr. Pitcher has put forward an argument which runs something like this: Whereas human tuberculosis of bovine origin has fallen, between 1945 and 1948, by about one half in Scotland and to a slightly less extent in England and Wales, the incidence of human pulmonary tuberculosis rose in Scotland and fell in England and Wales. The rise in human pulmonary tuberculosis in Scotland is due to the greater proportion of attested cattle. The milk of tuberculous cattle contains "tuberculin" which withstands pasteurisation and protects people against human pulmonary tuberculosis. Tuberculous mastitis should be eradicated by regular quarterly clinical examination, attestation should be halted and tuberculin testing made illegal. Attested herds are full of mastitis and milk should continue to be pasteurised to protect the public from the ill-effects of such milk. Although attestation gets rid of tuberculosis all other diseases are commoner in such herds.

This argument has errors all along the line. Here are some of them:—

1. Since 1948 the mortality rate and number of notifications of pulmonary tuberculosis in Scotland have fallen, although attestation has progressed.

2. There is no evidence that "tuberculin" is present in the milk of tuberculous cows. If it were present it would be unlikely to be absorbed, except from the intestine of the newborn infant. If it were absorbed there is no reason to think it would confer immunity. If tuberculin in milk were of value

it would be simpler and safer to add it to milk, before or after pasteurisation.

3. Clinical examination of cattle cannot effectively detect tuberculous mastitis. Some cases escape such examination.

4. What is the evidence that diseases increase in incidence in attested herds as a result of freeing them of tuberculosis? When you rid man or animals of a serious disease many of them survive longer and eventually contract another. Instead of dying at 30 from small-pox we may live to 70 and die of heart disease or cancer. Getting rid of one disease also allows attention to be focused on others not previously recognised. Changes in feeding, management and breeding affect the incidence of disease.

Mr. Pitcher is concerned at the rise in the incidence of human pulmonary tuberculosis between 1945 and 1948 in Scotland, and rightly so. He also gives figures from other countries which show such a rise and in which cattle have been almost freed from tuberculosis. He assumes this to be a case of cause and effect but does not advocate the drinking of tuberculous milk to give a primary tuberculous lesion and protection to subsequent infection with the human bacillus. On the other hand, Mr. Rainey thinks it is the bovine tubercle bacillus and not tuberculin in milk which gives immunity, and I agree. It may well be that young adults given pasteurised or attested milk and with no other access to bovine tubercle bacilli will be more liable to suffer from tuberculosis caused by the human bacillus. Without the bovine bacillus there is only the human one left to deal with and so all cases of tuberculosis in man will then be due to the human type. Deaths, disablement and the risk of endogenous reinfection from "healed" bovine primary lesions will have disappeared. Is not that a good thing? Should we not tackle human tuberculosis in this country as effectively as we are at last tackling bovine tuberculosis? In the United States some 80 to 90 per cent. of young adults now fail to react to tuberculin: this used to be the percentage which reacted. Some sanatoria are closing down for lack of patients. Such tuberculin negative adults would doubtless prove very susceptible in our own environment, which is still well supplied with human tubercle bacilli but, thanks to pasteurisation and tuberculin testing, less infected with the bovine bacillus. That their young adults are now fully susceptible to tuberculosis is a tribute to the action taken against tuberculosis in the United States by veterinarians and doctors alike. We shall presumably progress along the same path. Until we have sufficient sanatoria and more time and money to devote to detecting and controlling human tuberculosis we may need to use B.C.G. for children in heavily infected surroundings, but this is only a poor substitute for eradication. Our aim should be to protect the human population from bovine tuberculosis by making milk safe and by preventing direct transmission from infected animals. That is our contribution to human health. The human tubercle bacillus has to be controlled by public health measures outside our sphere.

If bovine tuberculosis is a valuable source of protection against infection of man with the human tubercle bacillus, such protection comes from primary infection acquired through raw tuberculous milk or from contact with infected animals. The results of such protection would not be shown for many years—until children fed on pasteurised or attested milk reach adult or middle age. If a rise in human tuberculosis were caused by failure to supply bovine primary infections the rise would occur many years after such failure. A rise in deaths or notifications of human tuberculosis in Britain to-day, if it were due to such a cause, would be attributable to drinking pasteurised milk over the last 20 years and not to the recent increase in attested herds. Whether human tuberculosis would increase from this cause I do not know. If there is a serious risk of this occurring the remedy is to attack human tuberculosis and not to abolish pasteurisation or stop attestation.

Quite apart from its value to human health, the eradication of bovine tuberculosis will improve the health and productivity of dairy cattle, reduce tuberculosis in pigs and dogs and eliminate it in cats. Meat inspection will be a much smaller task and much valuable meat will be saved. It is as a measure to improve the health of livestock that attestation is being carried out.

If it were certain that bovine tuberculosis in man gave a valuable protection against human tuberculosis to those who did not die or become disabled as a result, it would still be bad policy deliberately to abandon attestation. If everyone could be given mixed raw milk containing a dose of living tubercle bacilli just sufficient to give a primary tuberculous lesion and some immunity but without the risk of death or disablement and with no chance of later recrudescence to provide an endogenous reinfection disease, there might be some advantage in it. But there is no such possibility. The risk is too great, the price too high. Looked at as a premium to insure against human tuberculosis, the cost of drinking tuberculous milk is far too high. Pasteurised tuberculous milk is just infected milk rendered safe by heat, neither dangerous nor protective, and not a food to be recommended. One might as well eat cooked tuberculous meat or offal.

The position is this: we must press on with attestation and see that there are sufficient staff to ensure that attested herds can be kept free of the disease by tuberculin tests at proper intervals. The general health of dairy stock will improve and meat and milk will be saved. Having got rid of tuberculosis we must devote our efforts to those other diseases which eradication of tuberculosis leaves behind, some of them still little understood.—Yours faithfully, E. G. White, Clifton Lodge, New Chester Road, Hooton, Wirral, Cheshire. *October 12th*, 1951.

[This subject has been well ventilated in our columns and no further correspondence upon it can be considered after December 10th, 1951.—*Editor.*]

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## BLOOD SPLASHING OF MEAT

Sir,—Mr. Anthony's article is most interesting to the R.S.P.C.A., particularly in view of the exhaustive investigation that was carried out, on our behalf, into the cause of blood splash in pigs as long ago as 1932, in which Professor A. V. Hill, F.R.S., Professor B. McSwiney, M.B., B.CH., and Professor W. C. Clinton, M.I.E.E., played prominent parts.

Undoubtedly Mr. Anthony is right in that the lessening of the time gap between stunning and bleeding reduces splashing, but I wonder whether he can confirm the result of our investigations—namely, that lack of an adequate resting period after arrival in the slaughterhouse, especially in those cases where the pigs arrive in an overheated condition, or have been transported in very cold conditions, very materially increases the incidence of blood splashing.

The methods of slaughtering animals in the United States have caused grave concern to our sister organisations there for very many years and I am sending on to them copies of *The Veterinary Record* containing Mr. Anthony's article. I am sure that the author will be only too pleased to place the benefit of his knowledge and skill at their disposal in an attempt to ensure that, in future, humane methods are employed in the packing houses of America.—Yours faithfully, Arthur W. Moss, Chief Secretary, R.S.P.C.A., 105, Jermyn Street, London, S.W.1. *November 13th*, 1951.

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## BARRIER CREAMS

Sir,—As for nearly 100 years the arms of my grandfather, my father and myself have been successfully protected by a simple inunction of camphor, one part, dissolved in eight parts of olive oil, perhaps those who expose themselves to infection in calving and cleansing cows might like to know of this simple formula.—Yours faithfully, R. C. G. Hancock, Springhurst, Ecchinswell, Newbury, Berks. *November 16th*, 1951.

## DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

**Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases**

| Period | Anthrax | Foot-and-mouth | Fowl Pest | Parasitic Mange* | Sheep Scab | Swine Fever |
|---|---|---|---|---|---|---|
| Oct. 16th to 31st, 1951 | 7 | — | 43 | — | — | 97 |
| Corresponding period in— | | | | | | |
| 1950 | 12 | — | 4 | — | 2 | 32 |
| 1949 | 18 | — | 55 | — | — | — |
| 1948 | 9 | — | 2 | — | — | 1 |
| Jan. 1st to Oct. 31st, 1951 | 339 | 28 | 653 | — | 5 | 1,122 |
| Corresponding period in— | | | | | | |
| 1950 | 297 | 20 | 107 | — | 23 | 242 |
| 1949 | 195 | 13 | 510 | — | 34 | 5 |
| 1948 | 93 | 2 | 252 | 1 | 52 | 25 |

* Excluding outbreaks in Army Horses.

**Tuberculosis (Attested Herds) Schemes**

The number of Attested Herds, *i.e.*, herds officially certified as free from Tuberculosis as at October 31st, 1951, was as follows:—

| England | Wales | Scotland | Total (Great Britain) |
|---|---|---|---|
| 34,277 | 19,662 | 16,243 | 70,182 |